Chapter 42

DELAYED TREATMENT OF OTOLOGIC TRAUMA

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INTRODUCTION

The human ear is an exquisitely sensitive pressure transducer, able to defect minute changes in air pressure and convert them to neural signals for auditory perception. On the modern battlefield, numerous sources of impulse noise from weapons, continuous noise from equipment, and blast overpressure can damage this delicate sensory organ. As a result, hearing loss is a common injury. The biologic mechanisms leading to hearing loss from noise have been defined, and new therapeutic options are beginning to emerge based upon this knowledge. When auditory injury cannot be treated, hearing aids and other rehabilitative options can be used to minimize functional deficits.

The outer, middle, and inner ear are contained within the temporal bone, which comprises part of the skull base. In addition to injury resulting from battlefield noise, blunt force or penetrating trauma can result in fractures of this portion of the skull, leading to acute as well as long-term otologic injuries. Often these injuries can be managed without surgery. This chapter will review three areas of medical management. First, the biology of hearing loss from noise trauma will be discussed, as well as the current state of treatment in the acute setting. Second, a discussion of rehabilitative options for hearing loss, including unilateral deafness, will be provided. Third, the management of acute and chronic injuries from temporal bone fractures will be reviewed. Although these injuries are often addressed at Role 4 facilities due to the appropriate focus on more life-threatening conditions in theater, medical management for acute conditions of the ear can be undertaken at any role of care where adequate diagnostic and treatment capabilities exist.

ACUTE ACOUSTIC TRAUMA

Acute acoustic trauma (AAT) is defined as a sudden loss of hearing, partial or complete, caused by an extremely loud sound, a severe blow to the head, or other trauma. The greatest loss of hearing typically occurs at 4,000 Hz. It may be temporary or permanent. When of short duration, it is termed a temporary threshold shift (TTS). When recovery does not occur, the term permanent threshold shift (PTS) is used. Both duration and intensity of sound exposure contribute to the likelihood of either TTS or PTS developing. The Occupational Safety and Health Administration safe exposure limits are 8 hours for exposures of 90 dB of continuous noise and 140 dB for impulse noise. This standard is used to limit the amount of AAT from working in noise hazardous areas. Department of Defense and individual service guidelines are similar, with the caveats that operational considerations may necessitate that these exposure limits be exceeded. Noise from tracked vehicles, aircraft engines, weapons, and electrical generating or other equipment may easily meet or exceed these "safe" exposure limits.

AAT can manifest clinically as decreased hearing, tinnitus, aural fullness, and hyperacusis that presents immediately after a noise exposure above a critical value (which can vary from individual to individual).[1] Because the symptoms are identical at onset, only time and whether spontaneous recovery occurs can establish the distinction between TTS and PTS. Thus, presentation for medical care is typically delayed even in nonmilitary settings such as shipbuilding or other heavy industry where AAT occurs. In a 1976 study of 443 soldiers symptomatic after AAT, those who were removed from further noise exposure had greater hearing improvement than those who continued to be exposed.[2] Therefore, when possible, removal from further noise is the best known treatment for AAT. The study provides clinical evidence that the cochlea has the potential for recovery after injury providing no additional injury occurs.

When multiple regression analysis is applied to individuals with AAT, several factors emerge as prognostic for recovery: time from injury to presentation, and degree of hearing loss as measured by pure tone or high tone averages.[3] No significant relationship to outcome existed in this analysis for age at injury, use of hearing protection, or drug therapy with steroids, vitamin B, or dextran. The reason for the lack of benefit provided by use of hearing protection may be that noise levels sufficient to cause injury can exceed the protective capacity of the devices worn. Even with 20 to 30 dB of protection, sustained continuous noise levels higher than 110 dB or impulse noise levels over 160 dB are not uncommon on the battlefield. In fact, at these levels direct sound transmission through the body via bone conduction to the cochlea is a significant source of noise exposure, and injury can occur despite protection. Thus, while mechanical protection is necessary, it may not be sufficient. However, hearing protection remains the mainstay of prevention of AAT by preserving hearing in those who would otherwise suffer injury.

In those with hearing loss from AAT, only a small number will recover. In a 2008 study, only 21% of patients with an AAT completely recovered their hearing,

while 54% demonstrated partial recovery (though still below the normal range), and 25% had no recovery.[4] Those who presented later had a greater rate of unchanged hearing, further indicating the negative effects of prolonged noise exposure. Additionally, recovery was best predicted by the changes that occurred at 4,000 Hz. A return to normal at that frequency is the best predictor of eventual full recovery.

Biology of Noise-Induced Hearing Loss

While high-intensity sound exposures may cause direct mechanical damage to the delicate inner ear hair cells, much of the injury leading to both TTS and PTS is now known to result from damage caused by oxidative metabolism within the cochlea. Building upon earlier work, the mechanisms of inner ear injury from noise became well defined in the 1990s.[5,6] These pathways, both in the cytoplasm and the mitochondria, primarily occur in the metabolically active outer hair cells and provide multiple locations for pharmacologic intervention to prevent and treat cellular injury from noise toxicity. Noise-induced cellular injury may also cause reductions to cochlear blood flow and glutamate excitotoxicity.

The best studied compounds for cellular protection and recovery from noise injury are *N*-acetylcysteine (NAC), methionine, acetyl-L-carnitine (ALCAR), and ebselen.[7,8] NAC and methionine are both glutathione-replenishing agents. Glutathione is a natural intracellular antioxidant and free radical scavenger. Both agents provide increased antioxidant protection to cells during metabolic hyperactivity. ALCAR stabilizes mitochondrial action and prevents its disruption from over-activity. Ebselen is a novel chemical compound that serves as a catalyst for glutathione reductase and thus increases the intracellular concentrations of reduced glutathione to scavenge free radicals, thereby preventing oxidative injury. NAC, methionine, and ALCAR have all been shown in animal models to reduce the degree of PTS, either singularly or in combination, from AAT. However, ebselen is unique in that it has been shown to reduce TTS in addition to PTS.[9]

Glucocorticoid receptors are also known to localize to multiple areas within the inner ear, including hair cells, spiral ganglia, and other locations. Steroid receptor agonists in animals have demonstrated protection against noise injury, and antagonists provide the opposite effect. The exact mechanisms for agonists' protective effect are not entirely understood.[10] One thought is that glucocorticoids can improve cochlear blood flow. Reductions of cochlear blood flow and associated cochlear hypoxia have been shown to occur after acoustic injury.[11,12] As a result, hyperbaric oxygen has also been proposed as a treatment modality for AAT (see discussion below), even though this appears counterintuitive to the oxidative metabolism mechanisms.

Individual genetics also appear to play a role in susceptibility to noise injury and AAT. Individuals with essentially the same exposures and mechanical protection manifest differing degrees of hearing loss both acutely and over time. In a recent review of literature, the authors focused on human epidemiologic studies examining oxidative stress genes, inner ear potassium recycling pathway genes, and monogenic deafness genes.[13] Several promising genes were identified. Single nucleotide polymorphisms in these genes, if found to be predictive of susceptibility, may lead to new testing or treatments. Similar work in animal exposure studies has identified expression of metalloproteinases as important in the cochlear response to noise injury.[14] One of these studies points to a steroid responsive transcription factor that may help explain the beneficial effect of steroids.[15] Individual variations in this or similar genes and their expression may also account for the variability in response to treatment with steroids.

Clinical Studies of Acute Acoustic Trauma Therapy

Despite this wealth of basic science knowledge about the mechanisms of AAT and the promising animal studies on various pharmacologic interventions, human studies demonstrating efficacy of these therapies are extremely limited. Logistical impediments such as access to appropriate populations, identification of injury and uniform testing after injury (either self-report or mass screening), and individual variations in hearing protection use and susceptibility, as well as inadequate historical controls, have all proven significant limitations. As a result, there are currently no large-scale published studies in military populations, though several small-scale limited studies have been either completed or are underway, and some suggest benefit from medical intervention.

In a small, 53-subject, placebo-controlled cross-over study of male factory workers, *N*-acetylcysteine at a dose of 1,200 mg / day for 14 days was shown to statistically reduce TTSs.[16] These effects were most prominent in those with a particular genotype, further reinforcing the genetic aspects of noise-induced hearing loss and AAT reviewed previously. A subsequent, larger study of 363 subjects demonstrated similar effects.[17]

Given the effects of noise on cochlear blood flow, hyperbaric oxygen has also been examined in the treatment of AAT. In a comparison study evenly divided between hyperbaric and normobaric oxygen therapy

in 120 ears affected by AAT, hyperbaric oxygen resulted in nearly double the normal hearing recovery rate (70% vs 40%), though timing and duration of treatment were poorly controlled.[18] However, no control group without oxygen therapy was included in the study; such a group may have demonstrated an even greater benefit to supplemental oxygen over natural recovery.

Use of transtympanic injection of medications has also been explored to determine if this method has increased effects over oral dosing alone. In those with noise-induced hearing loss from AAT, a study examining concurrent transtympanic and oral steroid administration demonstrated double the hearing recovery rate in the combined therapy versus oral delivery alone (52% vs 23% for pure tone average, and 67% vs 31% in speech discrimination scores).[19]

Although these limited studies have demonstrated benefit from pharmacologic interventions in hearing recovery from AAT, the best choice of drugs, dosage, and duration of therapy remain to be determined. Additionally, all these studies suffer from differences in design, outcomes measures, and small subject numbers.

Timing of intervention has a significant impact on results as well. As would be predicted from the basic science studies, earlier intervention in humans also appears to have greater efficacy than delayed treatment. This can further complicate design and execution of large-scale studies. In a 2008 study by Psillas et al, soldiers exposed to rifle fire were divided into three groups according to treatment timing: (1) those treated in under 1 hour after injury, (2) those treated between 1 and 16 hours after injury, and (3) those treated over 16 hours after injury. Steroids and piracetam (a cyclic derivative of gamma amino butyric acid) were used for treatment. The group receiving treatment within 1 hour had five times the complete hearing recovery rate as those with the most delayed treatment (65% vs 13%).[20] In another study looking at tinnitus recovery after AAT, combinations of steroids, B vitamins, and trimetazidine (a fatty acid oxidation inhibitor used in angina pectoris) were used as treatment. Those receiving earlier intervention were predisposed to a better prognosis, although no differences between treatment groups were observed.[21] This may have been because the subjects' initial treatment ranged from 5 to 88 days after injury. Based on the data from the Psillas study, even those treated earliest in this time range were unlikely to demonstrate recovery, so the lack of difference may result from the delayed treatment rather than agents used. Thus, from the basic science work and these limited studies, earlier intervention appears to provide greater benefit regardless of treatment used.

Special Considerations in Acoustic Trauma From Blast

From the earliest medical descriptions of "shell shock," hearing loss and tinnitus have been among the most commonly cited initial complaints of blast-related trauma.[22] The condensed air pressure of the primary blast wave carries more energy than normal sound waves and thus can result in a greater degree of acoustic injury than would be predicted by sound intensity levels alone. The ear is particularly sensitive to this form of damage, and the head and neck have a higher rate of injury than areas of the body protected by armor.[23] Injury to the ear, particularly injury without visible damage such as hearing loss, is often missed in initial triage and care, when more life-threatening injuries are addressed. A review of expeditionary medical encounter data on 3,981 casualties who survived a blast showed that 1,223 (31%) were diagnosed with an ear-related injury at some point during the continuum of care in the first year after injury.[24] Thus, injuries to the ear and damage to hearing are among the most common injuries after blast, if not the most common.

Even before the widespread use of body armor during the first Gulf War, isolated ear injury from blast was recognized and reported.[25] Additionally, compared to prior conflicts, the increased protective effects of body armor have made previously fatal secondary and tertiary blast wave effects survivable, resulting in increased prevalence of injuries to the extremities and head and neck. Using the Joint Combat Trauma Registry, researchers at the Naval Health Research Center reviewed 4,623 blast injuries between 2004 and 2007.[26] While the majority of recorded injuries were in the extremities (41.3%), the second most common were in the head and neck (37.4%). Although this study underscores the great degree to which the head and neck are subject to recorded injury, in primary blast injury there may be no apparent physical injury, and thus hearing loss from AAT will be underreported.

When full diagnostic audiologic evaluations were performed postdeployment, the rate of observed hearing loss was much greater than expected.[27] Of the 250 subjects studied, nearly half (49%) reported tinnitus after blast, suggestive of at least temporary hearing injury after AAT. One-third had sufficient exposure to develop tympanic membrane perforation. Thus, even in the absence of other injuries, accumulated primary blast trauma can result in cumulative hearing losses over time. Studies from the current conflict have reinforced the need for predeployment and postdeployment hearing screening and prompt evaluation of acute otologic injury.[28]

Further recognition of the impact of blast injury upon the ear with resultant AAT and reduced combat effectiveness led to a Joint Theater Trauma System Clinical Practice Guideline which was first published in 2007,[29] with the most current revision dated March 2012. According to this guideline, patients with reported hearing loss persisting beyond 72 hours after a blast injury or acoustic trauma should undergo a screening hearing test or audiogram to determine the degree of injury. Those with a threshold shift greater than 60 dB on three consecutive frequencies are recommended for evaluation out of theater, bypassing Role 3 facilities.

Although study results have varied on the usefulness of tympanic membrane perforation as a marker for traumatic brain injury (TBI), significant hearing loss can be associated with a higher incidence of cognitive, psychological, or other physical injuries.[30–34] In a study of veterans returning from Operation Iraqi Freedom, those with blast-related TBI demonstrated much higher rates of sensorineural hearing loss (68%) and tinnitus (38%) than those with non-blast-related TBI or veterans who sustained TBI before this period.[35] The authors concluded that, because of the high rate of returning service members with blast-related TBI, new strategies to address the diagnosis and management of hearing-related disorders are needed.

In a larger study of over 12,000 veterans diagnosed with TBI between 2007 and 2009 who were compared to a matched cohort of 9,100 without TBI, more than one-third (35%) of those with TBI reported dual hearing and visual impairment, and only one-quarter (24%) reported no hearing or visual impairments.[36] The remainder had isolated hearing (31%) or visual (10%) impairment. The findings further underscore the need for complete and comprehensive evaluations in those complaining of auditory or other deficits who have been exposed to blast, even without obvious signs of trauma. Although not specifically addressed in this study, an additional class of sensory disorder is balance disorders. Visual, auditory, and balance impairments may be recognized long after injury or exposure. Dual sensory impairments are common and can create additional challenges for rehabilitation.

AUDITORY REHABILITATION

When hearing loss is not recovered, the mainstay of auditory rehabilitation remains amplification through hearing aids. The goal of amplification is to provide audition for communication and environmental awareness in a variety of listening conditions and, increasingly, to connect to other electronic devices such as handheld phones. The primary way this is achieved is by improving the desired signal over background noise level. Even those with mild hearing losses may benefit from amplification depending on their communication requirements. Additionally, those with cognitive or other impairments related to brain injury, which can interfere with central auditory processing, may benefit more than would be predicted based upon audiometric data alone. An audiologist can perform additional testing to determine the need and potential benefit from amplification.

As with other electronics, the past decade has brought numerous advances in hearing aid technology through use of digital and wireless technologies.[37] These advances have reduced the devices' size and increased their performance and ease of use. Compression circuitry to maintain a consistently comfortable signal-to-noise ratio allows a variety of signal intensities to be presented without the need for volume adjustment. Although not new, directional microphones are another recent enhancement to hearing aids. Previously, these microphones had not been regularly offered in hearing aids because their detection algorithms were not sufficiently robust to allow proper determination of when to switch between omnidirectional and directional settings. These deficiencies have been overcome, and although useful in only about 25% of listening situations, they can be very helpful when needed.[38] Digital technology has also allowed enhanced frequency response as well as sophisticated frequency transposition to provide sound information from frequencies that cannot be amplified in severe hearing loss.

Open-fit devices are another important advancement that utilizes the acoustic properties of sound in addition to modifying the signal electrically. Rather than using an occlusive ear mold, open-fit hearing aids have a small flexible tube connected to a small dome within the canal. This allows low frequency signals to bleed off, providing better boost to high frequency sounds, which are typically lost in AAT. Equally if not more important, the open fitting eliminates the occlusion effect from tight ear molds, greatly enhancing comfort for the user. What makes this open fit possible is the introduction of digital multichannel signal processing systems. These circuits can recognize and shut off a channel before creating feedback or introduce a 180° out-of-phase signal to cancel the feedback signal.

The Federal Communications Commission has mandated that hearing aids be compatible with standard and mobile phones. Bluetooth connections, while not incorporated into the actual device itself, are also

available as an accessory interface between hearing aids and communications devices. Options also exist to use smart devices for remotely controlling the hearing aid. (The original FCC regulation was adopted in 1988 and later modified in 2003 with requirements that mobile headsets abide by American National Standards Institute standard C63.19.) Despite these advances, the human ear and brain remain much more effective in detecting and localizing signals and separating them from noise. Thus, even with quality amplification, communication in the presence of noise may remain difficult for those with hearing loss.

In some circumstances, traditional hearing aid amplification will not result in benefit. Sufficient cochlear reserve, both for sensitivity as well as clarity, must exist. When this is not present, other strategies can be used to rehabilitate hearing. With severe to profound loss of hearing in one ear, the term single-sided deafness (SSD) is used to represent this special rehabilitation challenge. The lack of binaural input from SSD leads to a loss of spatial localization as well as significantly reduced discrimination in background noise, both of which are important elements of normal hearing. Normal binaural input allows for central auditory processing, which uses timing and frequency differences to provide localization as well as suppression of signal to improve perception in noise. Thus, SSD represents a sensory deficit. Those affected with SSD must learn to hear in another manner. Compensation mechanisms include avoiding adverse hearing environments (background noise, multiple speakers), and using head movement to detect acoustical differences. This type of sensory loss often results in isolation, anxiety, and even depression, to a greater degree than with other kinds of hearing loss.

The traditional methods for rehabilitating SSD have been contralateral routing of signal (CROS) and binaural amplification with contralateral routing of signal (BiCROS) devices. In a CROS system, a microphone is placed on the deaf side that transmits signal to the normal hearing ear via a wireless transmitter. In BiCROS systems, used for those with a hearing deficit in the only hearing ear, the same setup is used, but the signal is also amplified in the hearing ear. Typically BiCROS systems are better tolerated than CROS systems because users tend not to favor the amplified signal in their normal hearing ear.

Bone conduction provides an alternative methodology to provide this signal in those with SSD. Using the bone integration technology developed for dental and other prosthetic implants, bone-anchored hearing devices were first developed in the early 1990s in Europe. With approval by Medicare and other US insurance carriers in 2005, the use of these devices became widespread and a variety of options now exist. These devices transmit the signal from the deaf side to the intact cochlea via vibration of the skull, thus avoiding interference with the normal signal to the intact ear. The first and most commonly used of these devices is the bone-anchored hearing appliance (BAHA), which uses a hearing aid attached to a percutaneous bone implant placed above and behind the affected ear.

In a metaanalysis of studies comparing CROS to BAHA devices, BAHA seemed to provide improved auditory localization and speech discrimination compared to the CROS hearing approach. This finding has lent strong support for the use of BAHA.[39] However, these studies had multiple shortcomings including selection bias, low power, lack of randomization, and lack of hearing impairment stratification before selection for the studies. As a result, other studies have suggested that the data is inadequate to conclude whether a clear benefit exists for BAHA or CROS rehabilitation of SSD.[40] Examinations of the long-term use and benefit of BAHA by patients have also been undertaken to determine the value of these devices.[41] Of 56 patients with an average of 3.2 years of use, 81% continued to use the device. In addition, both the Glasgow hearing aid benefit profile and the abbreviated profile of hearing aid benefit demonstrated statistically significant improvement from baseline measures, though the benefits tended to decline over time. In this same study, 38% of the group experienced severe local skin reactions around the post site, but only one person required device removal.

This high rate of local skin reaction is not uncommon and has led to changes in operative technique and the development of other bone conductive devices. Sonitus Medical (San Mateo, CA) is the maker of the SoundBite, a device that uses a dental retainer to provide sound transmission from a wireless ear-level microphone. Its chief advantage is that it does not require surgery. However, its power is limited, it is unable to provide a full day of use, and it cannot be worn while eating. Another option are the Alpha 1 and Alpha 2 devices made by Sophono (Boulder, CO) that use an implanted magnet (MRI compatible) to transmit bone conduction without the use of a percutaneous post. This avoids the problems associated with skin reaction at the cost of reduced signal gain. The most recently approved device is a hybrid of the traditional BAHA and the Sophono. The BAHA Attract system produced by Cochlear Ltd (New South Wales, Australia) uses the traditional osteo-integrated post, but rather than connecting transcutaneously, a magnet is attached to the post to receive the transmitted signal from the microphone and amplifier. All these newer devices also come with enhancements

to the external processors, including dual microphones to improve hearing in environments where background noise is an issue, and integrated links for radios, personal music players, cell phones, and similar items. This integration allows the use of these communications without sacrificing ambient listening in the normal ear.

Despite these advancements and the improved devices, bone conduction for SSD does not appear to restore normal binaural hearing. In the normal state, auditory signals arrive at individual ears with timing, phase, and frequency differences. These differences are integrated within the brainstem and central auditory processing to provide range, location, and azimuth as well as signal-to-noise discrimination and the ability to switch from one signal to another in the presence of noise. In SSD, when rehabilitation is provided via bone conduction or CROS, only one cochlear signal is present, and these binaural benefits are not obtained. Electrophysiologic studies of brainstem and long-latency auditory evoked potentials have been performed to determine what happens centrally to the bone-conducted signal in this pseudo-binaural state. As expected, the brainstem waveforms between the two signals are delayed due to the short time delay between the bone-conducted and normally conducted signal. However, the longer latency responses reflecting central cortical processing demonstrate no differences.[42] As a result, when studies of localization and signal discrimination compare subjects with normal hearing to subjects using BAHA, those with unilateral deafness demonstrate little to no improvement over the non-aided condition.[43] Thus, while bone-conducted devices may represent an improvement over CROS aids to eliminate head-shadow effects (when the good ear is "shadowed" from the signal by the head) they do not restore true binaural hearing for localization and signal discrimination. Caution must therefore be exercised in return-to-duty determinations for those rehabilitated in this manner.

Given that bilateral cochlear input is needed for true binaural hearing, recent attention has turned to the use of cochlear implants for SSD. Traditional thinking has been that with normal contralateral hearing, the signal from a cochlear implant would be too distorted to be acceptable. Tinnitus can arise from SSD due to the loss of auditory input and, in rare cases, this can be disabling. As a result, when other therapies failed, investigation turned to the use of cochlear implantation, not for hearing benefit, but to reduce tinnitus.[44] A significant benefit was obtained with these implants and the auditory signal was surprisingly well-tolerated. These results prompted further study of the demonstrated improvements in hearing in the presence of noise and subjective localization with the implant activated over the normal hearing ear alone.[45,46] Currently the literature contains 17 publications concerning a total of 108 total patients in case series, prospective, and retrospective studies.[47] Taken as a whole, the evidence to support the benefits for sound localization and discrimination with cochlear implants appear to be valid.[48] However, further study is required to optimize patient selection, signal processing strategies, and programming to maximize benefit with a contralateral hearing ear. In addition, newer devices with short electrodes to preserve remaining hearing and hybrid devices capable of both electric and acoustical stimulation are either available or undergoing approval.

BASILAR SKULL FRACTURE

Basilar skull fractures are a relatively uncommon result of blunt or penetrating head trauma, accounting for roughly 4% of all skull fractures. However, when basilar skull fractures do occur, some 75% involve the temporal bone and they often result in injuries requiring consultation with an otolaryngologist. Traditionally these fractures have been classified as longitudinal or transverse in relation to the long axis of the petrous pyramid. However, in many cases the fractures are mixed. Another method of classification is in relationship to the otic capsule, either sparing or violating. In one recent study, 45% of fractures were judged to be longitudinal, 29% transverse, and 26% mixed.[49] Using the alternative grading system, 93% spared the otic capsule while 7% violated the capsule. Regardless of the radiologic classification, the main complications to be evaluated are hearing loss, cerebrospinal otorrhea, and cranial nerve injury with particular attention to the facial nerve. In most cases these injuries can be managed conservatively, allowing for more serious and life-threatening injuries of the head and body to be addressed first. As a result, medical and surgical management will be deferred to higher roles of care, often occurring at facilities providing definitive care.

Hearing loss can be conductive due to fluid or blood in the ear canal or middle ear, or sensorineural from concussive effects on the labyrinth or direct damage due to fracture of the otic capsule. Early physical examination and the use of tuning forks can often determine the nature of the hearing loss. Horizontal fractures and those violating the otic capsule carry a higher risk of sensorineural injury. High resolution temporal bone scans may demonstrate a disruption of the ossicular chain at the joint of the malleus head and

incus body, or incus and stapes. Definitive audiologic evaluation can be deferred until middle ear fluid or blood has resorbed and spontaneous healing of traumatic tympanic membrane perforations has occurred. Typically this is 60 to 90 days postinjury. If significant sensorineural loss is suspected from the initial physical examination, treatment similar to that for AAT (10–14 days of high dose corticosteroids) can be attempted provided no other medical contraindications exist. Medical treatment is not recommended for obvious disruptions of the cochlea or labyrinth.

Cerebrospinal fluid (CSF) otorrhea can occur in a significant fraction of basilar skull fractures, as often as 15% of cases in one large series.[50] When clear otorrhea is present in the setting of a temporal bone fracture, CSF should be suspected. While highly specific tests for CSF such as beta-2 transferrin are available, these often require a relatively large quantity of fluid (several milliliters) and can take weeks for results. A more practical bedside test is known as the "ring" sign, in which a drop of the fluid is placed on a piece of filter paper. Other absorbent media may also be used including tissue, paper towel, or bed linens. A halo of clear fluid will surround any blood that remains in the center. Unfortunately, this test, while simple and quick, is not specific. Saline, water, and nasal secretions when mixed with blood will all result in a ring sign.[51] These other fluids are unlikely to be present in the ear and, while the test may have a high false positive rate for CSF rhinorrhea, it can be considered reliable in the case of CSF otorrhea.

Over the years the use of antibiotic prophylaxis in the setting of CSF otorrhea has been controversial in the neurosurgical literature. In a 1996 study, rather than reducing the rate of meningitis, the use of prophylactic antibiotics carried a significantly higher incidence of the development of meningitis.[52] The current recommendation for basilar skull fractures based upon a Cochrane review is not to use antibiotic prophylaxis for CSF leak.[53] Instead, treatment is bed rest with head elevation, stool softeners, and avoiding increased intracranial pressure from straining, cough, or sneezing with a closed mouth. With these conservative measures the majority of these leaks will spontaneously resolve within 1 week. For those that do not resolve, lumbar drainage or surgical repair can be considered because the risk of meningitis increases 8-fold when the duration of leak exceeds 7 to 10 days.

Given its long course through the temporal bone, the facial nerve is at particular risk of injury in temporal bone fracture, either from direct transection, stretch, or swelling. The majority of patients with these injuries will spontaneously recover, with severity and time of onset as the two most important prognostic factors for outcome. If the endoneural tubules remain intact, Sunderland's classification of axonal injury predicts complete recovery.[54] If the onset of complete paralysis is delayed, then the endoneurium can be considered intact. The outcome is universally favorable for normal or near normal recovery and surgical decompression is not indicated. Steroids may be given to reduce edema if not otherwise contraindicated due to other injury. Likewise, if there is paresis only and no complete paralysis, prognosis is excellent with steroid use and observation alone. Recently, calcium channel blockers have been studied to enhance regeneration of damaged motor nerve fibers. Animal studies demonstrated nimodipine was able to: (*a*) enhance axonal sprouting after nerve repair and crush injuries; (*b*) accelerate the time course of functional recovery and axonal regrowth; and (c) increase numbers and sizes of myelinated axons in the facial nerve.[55,56] These promising results led to a small pilot study of 13 patients who developed moderate to severe facial paresis after maxillofacial surgery.[57] As with the animal studies, earlier than expected recovery was obtained with House-Brackmann grades 1 to 2 results within 2 months.

With unconscious patients, examination of facial function will be more difficult. Painful stimuli may be used to elicit a grimace. Alternatively, nasal flair may be observed with normal respirations. Tightly pinching the nasal ala for 1 minute and then releasing may also be used to observe for a reflex contraction. If no physical sign of facial function can be observed, electrical stimulation can be used. Either a Hilger unit or an anesthesia nerve stimulator can be used to stimulate the main trunk of the facial nerve at the angle of the mandible. This must be done no earlier than 72 hours postinjury to allow wallerian degeneration from a potential more proximal injury to manifest. If no movement with electrical stimulation is obtained, surgical exploration and decompression should be considered. In unilateral fractures, electroneuronography (ENoG) will provide the most accurate assessment, but it is not always available. For those with over 90% degeneration on ENoG, surgical decompression is indicated. If a clear site of injury is found, then that nerve segment should be widely decompressed and any bony fragments removed. Incomplete sections less than 50% of the nerve width should also be widely decompressed. For injuries larger than this or complete transection, a longer segment of nerve can be mobilized for direct anastomosis or cable graft if insufficient length for a tension-free anastomosis is available after mobilization. Earlier repair will lead to better outcomes. In

a study of 66 patients with complete paralysis who underwent facial nerve decompression after temporal bone trauma, those with decompression within 3 weeks had a 93% House-Brackmann grade 1 or 2 (85% grade 1) result. Those decompressed after 2 weeks but before 2 months had only a 70% grade 1 or 2 result (40% grade 1) while those treated after 2 months had only 40% grade 1 or 2 outcomes (15% grade 1), which approaches results from observation alone.[58] Thus, decompression optimally should be performed within 2 weeks of injury. When surgery cannot be done safely within 2 months, surgery is unlikely to improve outcome and continued observation should be strongly considered.

SUMMARY

Military operations are an incredibly loud environment. Auditory injury from AAT has been and will continue to be common among service members. Hearing protection devices can mitigate this rate to some extent. However, the sound intensity levels may exceed the protective capacity of the devices, and hearing may not always be protected due to unexpected loud exposures such as blasts. Current standard medical treatment consists of removal from additional acoustic trauma and treatment with oral and intratympanic steroids. Several additional agents are currently under study that hold promise to provide additional protection from noise as well as recovery from acoustic injury. As these products become approved for use, the best resource for up-to-date knowledge will be the recently established Defense Center of Excellence for Hearing in San Antonio, Texas. In particular, the Pharmaceutical Interventions for Hearing Loss working group will have knowledge of the latest developments underway.

Numerous advances have recently been made in the area of hearing rehabilitation. New devices and indications are emerging every year and the pace of technological advancement in this area is unlikely to decline. Improved traditional hearing amplification as well as implanted devices will expand our ability to adequately rehabilitate hearing loss to approach normal functional capacity. While the emergence of new pharmacologic treatments may mitigate the incidence and severity of hearing injuries, they will continue to occur. Thus, collaboration with audiologists familiar and skilled in the latest rehabilitative options is an essential role of the treatment team.

Temporal bone trauma from blunt or penetrating injury may lead to numerous sequelae such as CSF leak, hearing loss, and facial paralysis. Fortunately, these conditions frequently respond well to medical management. In cases where more definitive intervention is required, the optimal treatment window allows for more serious injuries to be stabilized or the patient to be transferred to higher roles of care where more specialized treatment is available. As with advanced treatments for auditory rehabilitation, this knowledge base and skill set will be concentrated at facilities offering definitive care.

CASE PRESENTATIONS

Case Study 42-1

Presentation

A 45-year-old woman fired a .45 caliber handgun without hearing protection. Upon the first shot, immediate pain, fullness, tinnitus, and decreased hearing were noted in both ears, but more so in the left. She was referred to an otolaryngology/head and neck surgery clinic 2 weeks after the incident because her symptoms were persistent on the left side. She now was also complaining of hyperacusis on the affected side.

Findings and Treatment

Otologic exam was normal except for tuning fork lateralizing to the right. Fistula test was negative. Audiometric testing demonstrated a left sensorineural hearing loss with speech reception threshold at 50 dB, compared to 20 dB on the right, and a poor word recognition score of 36% (Figure 42-1). High dose oral steroids (prednisone 60 mg/day) without taper were given for 2 weeks. Transtympanic steroid injection was offered, but declined. Repeat audiologic examination demonstrated minimal improvement in the speech reception threshold, at 40 dB, but word recognition improved substantially to 72% (Figure 42-2). Symptomatically, tinnitus, fullness, and hyperacusis were all unchanged. Referral for hearing aid evaluation was made, but fitting was difficult due to the hyperacusis despite the hearing loss being otherwise amenable to amplification.

Lessons Learned

Even seemingly "safe" firearms produce a significant amount of noise exposure. The standard .45 caliber round produces 157 dB at the muzzle. This

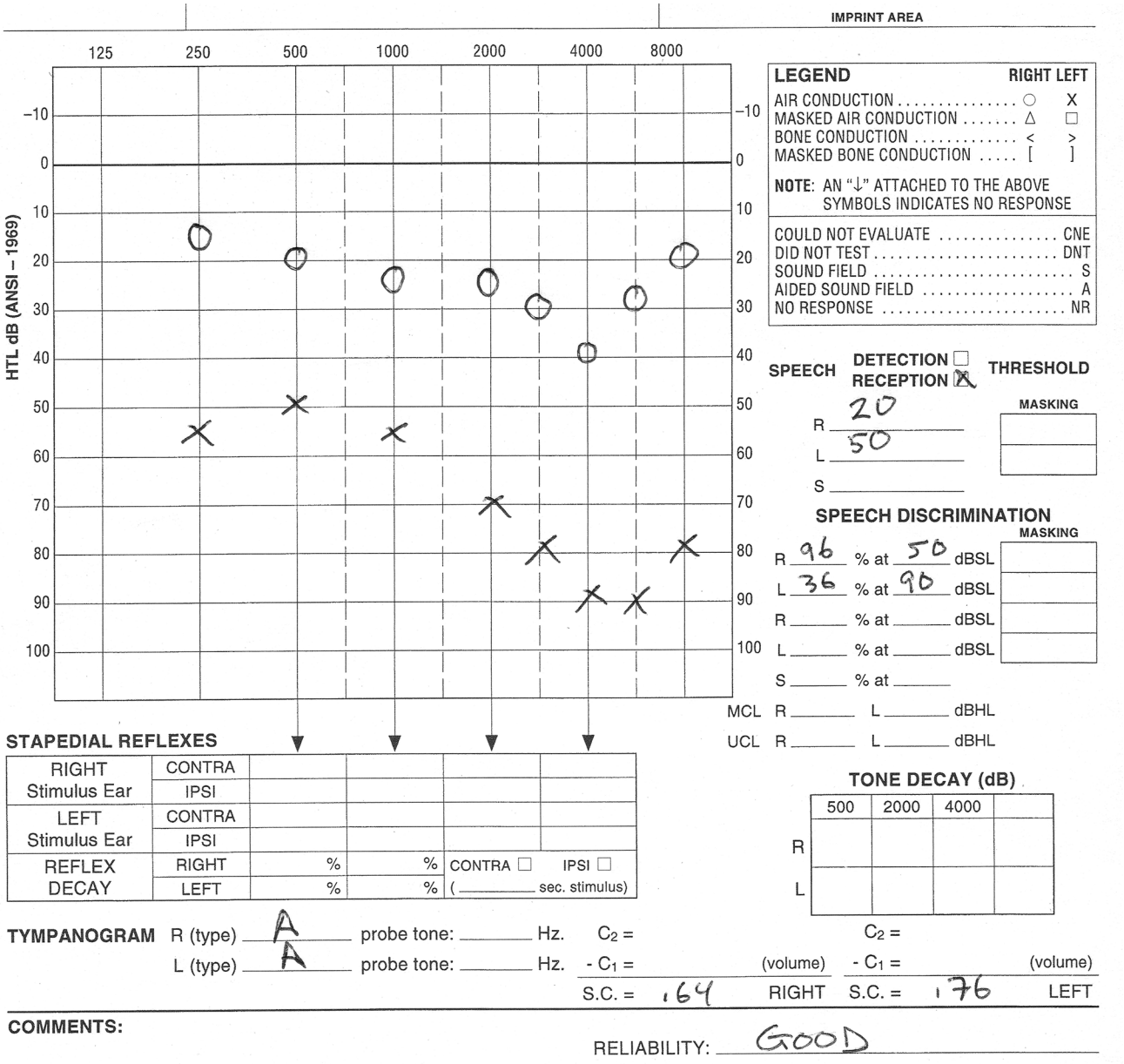


Figure 42-1. Audiogram at initial presentation after acute acoustic trauma. Note the loss at 4 kHz, which is a poor prognostic sign for recovery.

sound intensity to the unprotected ear is expected to produce at least temporary and possibly permanent hearing loss from AAT. Hearing protection during firing is crucial to prevent these injuries. Due to a head shadow effect, the left ear will be typically more affected than the right in right-handed shooters, as is seen in this case. Treatment with oral steroids alone offers limited benefit; they are more beneficial if given within hours to days rather than weeks after the injury. If benefit is obtained, it will typically be in improvement of word recognition scores as was seen in this case. This treatment can be important in creating the conditions for rehabilitation with hearing aids. Often, however, there is no appreciable improvement in symptoms such as fullness, tinnitus, or hyperacusis. These symptoms may decrease over time, but improvement typically occurs months to over a year. Rehabilitation can prove difficult in the face of these additional symptoms, particularly with a contralateral normal hearing ear.

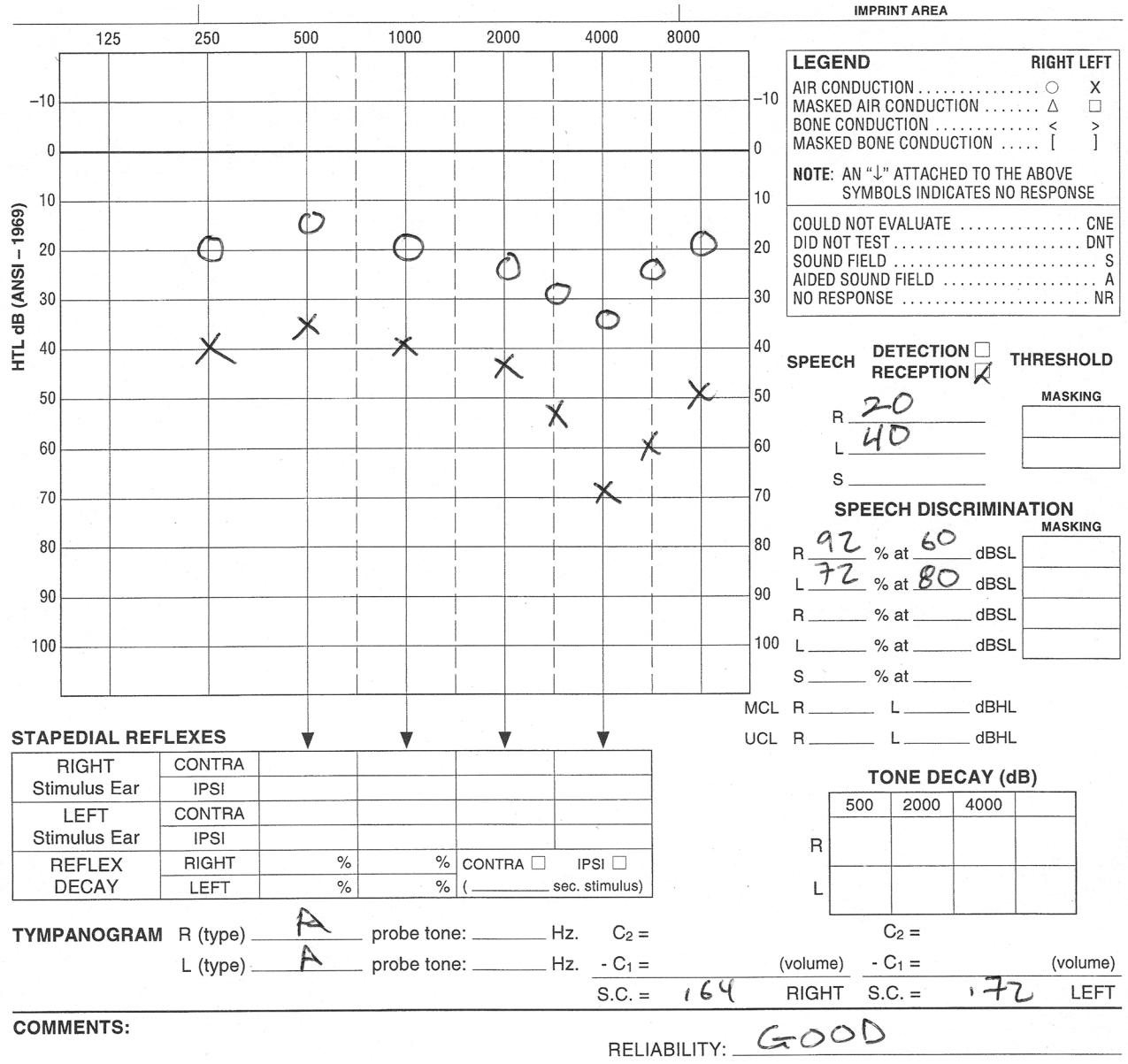


Figure 42-2. Audiogram after 2 weeks of oral steroid treatment. Although there has been some recovery in speech discrimination, making the use of a hearing aid as rehabilitation possible, the overall function remains less than optimal.

Case Study 42-2

Presentation

A 19-year-old man presented after sustaining a head injury and left temporal bone fracture after blunt trauma due to fall of 8 ft. He was hit on the right side of his face and fell and hit the left side of his head on the ground. Despite nausea, vomiting, and headache, he did not seek immediate medical attention. After sleeping, he awoke with left sided bloody otorrhea and sought care.

Findings and Treatment

He was evaluated with computerized tomographic imaging, which demonstrated three 8- to 14-mm areas of high attenuation in the right frontal lobe, likely representing a parenchymal contusion with hemorrhage, and areas suspicious for small subdural hematomas in the right middle and anterior fossa without mass effect or shift of midline structures. Additionally, there was a fracture through the temporal bone in the region of the mastoid process and air cells (Figure 42-3). There

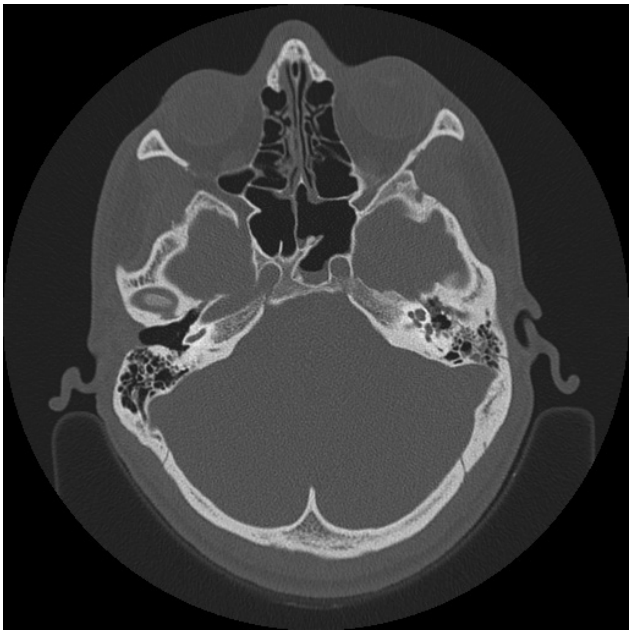
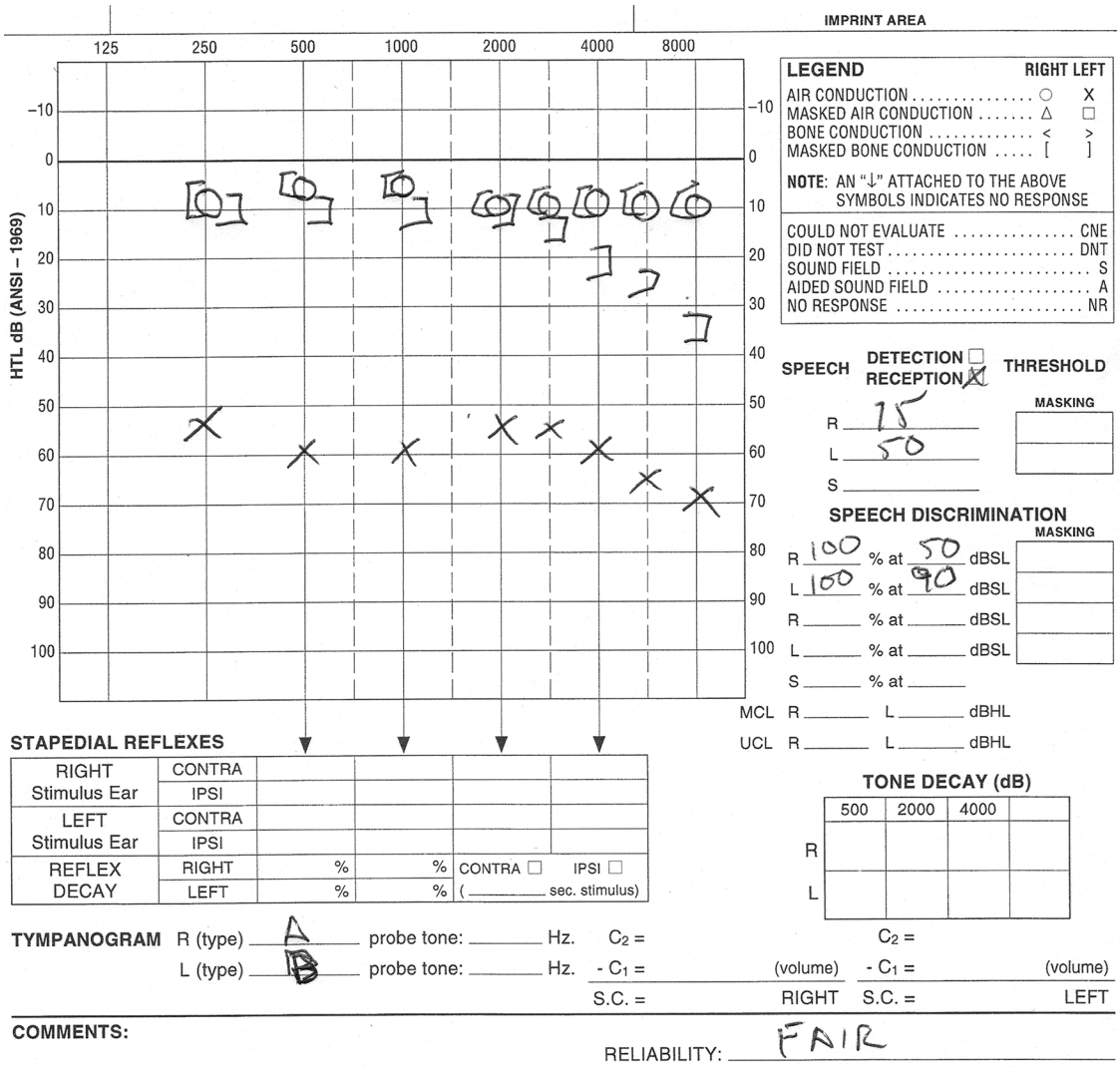


Figure 42-3. Computerized tomography scan demonstrating the fracture of the left temporal bone and fluid signal in the middle ear and mastoid air cells due to cerebrospinal fluid leak and blood. Also note the fracture in the lateral orbital wall.

was opacification of the mastoid air cells and middle ear cavity with fluid signal. The facial sinuses were well aerated. No fracture of the frontal bone, zygoma, or maxilla was seen.

The patient was admitted from the emergency room and otolaryngology and neurosurgery consultations were obtained. The neurosurgeon's recommendation was that if repeat imaging was unchanged overnight, the patient could be discharged. Otolaryngologic exam revealed bloody otorrhea and a tympanic membrane perforation on the left side. Also, the tuning fork lateralized to this side. Facial nerve function was intact.



The patient was sent home on stool softeners with instructions to remain at bed rest with his head elevated to resolve the CSF leak. He did not follow these instructions and presented again with continued left ear clear drainage, headache, and a mild left facial paresis. He was then admitted and placed on bed rest, head elevation, and observation. The facial weakness did not progress and the otorrhea resolved within 3 days.

The patient was seen again 1 month after injury, complaining of continued hearing loss. An audiogram was obtained that revealed a maximal left conductive hearing loss with speech reception thresholds of 50 dB and 100% discrimination with normal hearing on the right (Figure 42-4). Facial function was normal on physical exam at this time. An effusion was still present in the middle ear. Review of the imaging was suspicious for disruption of the ossicular chain at the malleus-incus joint (Figure 42-5).

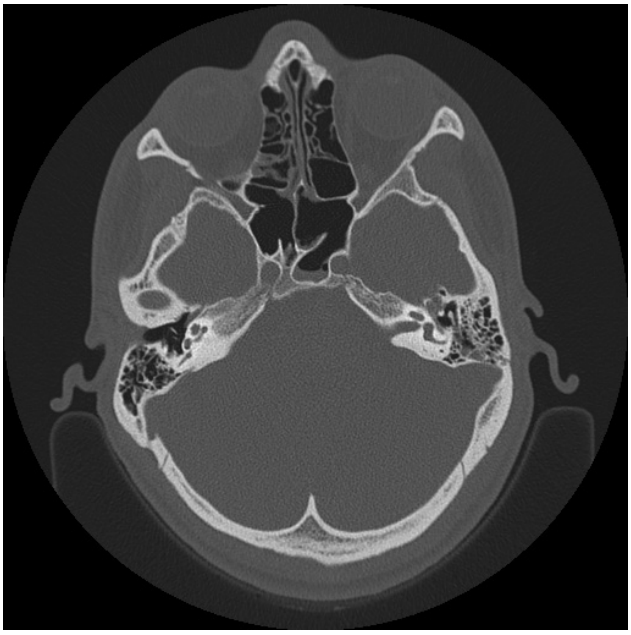


Figure 42-5. Computerized tomography scan at the level of the ossicular heads at the time of initial injury. Note the disruption of the malleus from the incus, confirming the ossicular discontinuity.

Lessons Learned

This case illustrates a delayed presentation of temporal bone fracture. Blunt head injury is common, and even patients with significant intracranial injuries may not present with symptoms prompting medical attention. Even with a temporal bone fracture and tympanic membrane perforation, several hours may pass before otorrhea will manifest. Thus, a high index of suspicion and careful examination are warranted. The failure to follow recommended limited activity likely led to the delay in resolution of the spinal fluid leak and increased the risk of development of meningitis. In less reliable patients or those who may not be able to keep duty restrictions, continued hospitalization for observation is recommended. As the facial paresis remained mild in this case, no steroids were given. If a delayed complete paresis developed, then steroids would have been given. The ossicular disruption was not noted on the initial review of the imaging. This is not uncommon in the face of multiple other intracranial or other types of injuries, which can be subtle. Hearing testing is typically deferred until any middle ear effusion has resolved, generally 2 to 3 months after injury. Here, the testing helped confirm the ossicular injury because the conductive deficit was greater than would be expected from the effusion alone. Surgical reconstruction can be deferred until any other injuries have stabilized.

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Figure 42-4 (*facing page*). Audiogram with maximal hearing loss pattern suggesting ossicular discontinuity rather than middle ear effusion alone as the cause of hearing loss.

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